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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/744,282	04/05/2001	Andreas Martinus Maria Miltenburg	0/98394US	4006

7590 10/21/2003  
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EXAMINER

HUYNH, PHUONG N

ART UNIT

PAPER NUMBER

1644

DATE MAILED: 10/21/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

<b>Office Action Summary</b>	<b>Application No.</b>	<b>Applicant(s)</b>	
	09/744,282	MILTENBURG ET AL.	
	<b>Examiner</b>	<b>Art Unit</b>	
	Puong Huynh	1644	

**-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --**  
**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE Three MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☒ Responsive to communication(s) filed on 21 August 2003.
- 2a) ☐ This action is **FINAL**.                      2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 4,6,10,12 and 16 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 4,6,10,12 and 16 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
- Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on \_\_\_\_\_ is: a) ☐ approved b) ☐ disapproved by the Examiner.
- If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

**Priority under 35 U.S.C. §§ 119 and 120**

- 13) ☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☒ All    b) ☐ Some \*    c) ☐ None of:
1. ☒ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- \* See the attached detailed Office action for a list of the certified copies not received.
- 14) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).
- a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

**Attachment(s)**

- |  |   |
|--|---|
| 1) <input type="checkbox"/> Notice of References Cited (PTO-892)                             | 4) <input type="checkbox"/> Interview Summary (PTO-413) Paper No(s) _____   |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)         | 5) <input type="checkbox"/> Notice of Informal Patent Application (PTO-152) |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ | 6) <input type="checkbox"/> Other:  |

#### DETAILED ACTION

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 8/21/03 has been entered.
2. Claims 4, 6, 10, 12 and 16 are pending and are being acted upon in this Office Action.
3. The disclosure is objected to because of the following informalities: (1) The US Pat App. No. 619,645 on page 1, line 2 should have been 08/619,645; (2) The (PTFGRSFTLASSETGVG) on page 7, lines 19-24 should have been (RSFTLASSETGVG). (3) the "effcet" on page 4, line 20 should have been "effect". Appropriate action is required.
4. Claims 6 and 12 are objected to because said claims depend from canceled claim 5.
5. The following is a quotation of the first paragraph of 35 U.S.C. 112:  

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.
6. Claims 4, 6, 10, 12 and 16 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling only for (1) a method of treating inflammatory rheumatoid arthritis by inhibiting the proliferation of autoreactive T lymphocytes and inflammation associated with rheumatoid arthritis, comprising the step of administering a pharmaceutical composition nasally comprising an effective amount of HC gp-39 or fragment thereof wherein the fragments are selected from one or more SEQ ID NO: 1-8 and a pharmaceutically acceptable carrier, **does not** reasonably provide enablement for (1) a method of treating *any* autoimmune disease such as rheumatoid arthritis by "**modulating**" the reactivity of lymphocytes associated with said disease, comprising the steps of administering a pharmaceutical composition comprising an effective amount of *any* HC gp-39 or fragments thereof wherein said fragments are selected from one or more of SEQ ID NO: 1-8 and a pharmaceutically acceptable carrier, wherein

said lymphocytes are reactive to *any* “antigens other than HC gp-39” which are present in the same tissue as HC gp-39 as set forth in claim 4, (2) a method for “modulating the reactivity of lymphocytes that are reactive to *any* antigens other than HC gp-39” which are present in the same tissue as HC gp-39, comprising the steps of administering a pharmaceutical composition as set forth in claim 10, and (3) a method of treating any autoimmune disease by modulating the reactivity of lymphocytes associated with *any* undisclosed autoimmune disease comprising the steps of administering a pharmaceutical composition comprising an effective amount of HC gp-39 or fragments thereof, wherein said fragments are selected from one or more of SEQ ID NO: 1-8 and a pharmaceutically acceptable carrier, wherein said lymphocytes are reactive to *any* antigens other than HC gp-39 which are present in the same tissue as HC gp-39. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in **scope** with these claims.

Factors to be considered in determining whether undue experimentation is required to practice the claimed invention are summarized *In re Wands* (858 F2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)). The factors most relevant to this rejection are the scope of the claim, the amount of direction or guidance provided, the lack of sufficient working examples, the unpredictability in the art and the amount of experimentation required to enable one of skill in the art to practice the claimed invention. The specification disclosure is insufficient to enable one skilled in the art to practice the invention as broadly claimed without an undue amount of experimentation.

The specification discloses only a method of treating inflammatory rheumatoid arthritis by nasal induction tolerance comprising the step of administering a pharmaceutical composition comprising an effective amount of bovine collagen type II HC gp39 (page 10) and a pharmaceutically acceptable carrier (page 14). The specification further discloses HC gp-39 induces the proliferation of PBMC in vitro obtained from some patient with rheumatoid arthritis (See page 15). The specification on page 8 further discloses that administering high or low doses of the tolerogen can attain tolerance or peptides and the amount of tolerogen or peptide will depend on the route of administration, the time or administration, the age or the patients as well as general health conditions and diet.

The specification does not teach how to treat *any* “autoimmune disease” such as osteoarthritis, alcohol-induced liver fibrosis, inflammatory bowel disease, and systemic lupus erythematosus (SLE) and rheumatoid arthritis by “modulating the reactivity of lymphocytes

associated with any "autoimmune disease" comprising the step of administering a pharmaceutical composition comprising HC gp-39 fragments thereof wherein the fragments are selected from one or more of SEQ ID NO: 1-8 and a pharmaceutically acceptable carrier and wherein said lymphocytes are reactive to "antigens other than HC gp-39" which are present in the same tissue as HC gp-39 for the following reasons. First, the term "modulating the reactivity of lymphocytes" encompasses stimulatory and inhibitory activities, which are mutually exclusive. It is not clear if the claimed method of treating any autoimmune disease including rheumatoid arthritis is to inhibit or to stimulate the reactivity of which lymphocytes. Second, there is insufficient guidance as to which "antigens" other than HC gp-39 that the isolated PBMCs from rheumatoid arthritis are reacted to. Third, Claim 16 encompasses treating any autoimmune disease by administering one or more HC gp-39 fragments selected from one or more of SEQ ID NO: 1-8 by modulating the reactivity of lymphocytes wherein the lymphocytes are reactive to any antigens other than HC gp-39 which are present in the same tissue as HC gp-39. Given the indefinite number of autoimmune disease, there is insufficient guidance and in vivo working example that the claimed fragments of HC gp-39 selected from one or more of SEQ ID NO: 1-8 would be useful and effective for treating *any* autoimmune disease. Even if the method is limited to treating rheumatoid arthritis, there is no in vivo working example using any HC gp-39 fragments, much less which lymphocytes are reactive to which antigens other than HC gp-39. Fourth, it is well known that induction of tolerance depends on a number of factors including the route of administration, dose, and etc as disclosed on page 8 of the specification. In vitro and animal model studies have not correlated well with in vivo clinical trial results in patients. Since the therapeutic indices of biopharmaceutical drugs and particularly tolerance induction can be species- and model-dependent, it is not clear that reliance on the limited in vivo experimental model accurately reflects the relative efficacy of the claimed method of tolerance induction regimens in treating all autoimmune disease that are mainly T cell mediated. Although in vitro and animal models validate concepts based on studies of human disease, such studies are limited to the acute as opposed to chronic nature of autoimmune disease.

Van Noort *et al.*, of record, teach autoimmune diseases can be species and model-dependent (See entire document, pages 167-168, in particular). Given the indefinite number of undisclosed inflammatory autoimmune disease, it is not clear that the reliance on the limited in vivo experimental model accurately reflects the relative efficacy of the claimed method of tolerance induction regimens in treating all autoimmune disease that are mainly T cell mediated.

Anderton *et al*, of record, teach peptide-based immunotherapy of autoimmunity is unpredictable and peptides that inhibit autoimmune disease such as encephalomyelitis *in vitro* actively induce disease *in vivo* (See page 370, column 1, second full paragraph, bridging column 2, first paragraph, in particular). Further, Anderton *et al* further teach that clinical trial was suspended due to hypersensitivity reactions in a significant proportion of patients (See page 370, column 2, second paragraph, in particular).

Verheijden *et al* (PTO 1449) teach tolerance can be attained by the amount of autoantigen administered and the **route** of administration is just as important as the autoantigen such as human cartilage glycoprotein-39 (HC gp-39) itself. Verheijden *et al* teach administering a single injection of HC gp-39 in FIA to female BALB/c mice induces clinical signs of arthritis (page 1121, column 2, in particular) whereas intranasal administration of HC gp-39 before immunization completely abrogated DTH response upon challenge (See page 1122, column 2, last paragraph, in particular).

The Merck manual, of record, does not recognize the use of *any* HC gp-39 fragments thereof for treating *any* inflammatory autoimmune disease such as rheumatoid arthritis (See page 420-421, in particular). Reasonable correlation must exist between the scope of the claims and scope of enablement set forth. Further, the term “modulating the reactivity of lymphocytes” is not sufficient to define the biological activity to which the HC gp-39 and fragments thereof modulates such as inhibit or stimulated said undefined reactivity.

With regard to lymphocytes are reactive to *any* antigens other than HC gp-39 which are present in the same tissue as HC gp-39, the specification discloses only lymphocytes are reactive to HC gp-39, the specification does not teach antigens other than HC gp-39 which are present in the same tissue as HC gp-39. Myers *et al*, of record, teach autoimmune response to collagen type II in the CIA models is complex, requiring specific major histocompatibility complex (MHC) molecules, collagen type II specific T cell and B cell immune responses and their associated cytokines (See page 1862, in particular). Myers *et al* teach although the use of altered peptides in the treatment of autoimmune disease such as rheumatoid arthritis is receiving considerable attention as the evidence of their efficacy continues to grow *in vitro* studies as well as in animal models. However, the development of such therapeutics for human diseases relies upon significant knowledge of the autoantigen (See page 1873, second full paragraph, in particular). Given the indefinite number of antigens other than HC gp-39, a person of skill in the art could not predict which particular antigen is essential and could be used in a therapeutic method of treating

any autoimmune disease, much less for treating rheumatoid arthritis. In vitro and animal model studies have not correlated well with in vivo clinical trial results in patients.

As such, it would require undue experimentation of one skilled in the art to practice the claimed invention. See page 1338, footnote 7 of Ex parte Aggarwal, 23 USPQ2d 1334 (PTO Bd. Pat App. & Inter. 1992).

In re wands, 858 F.2d at 737, 8 USPQ2d at 1404 (Fed. Cir. 1988), the decision of the court indicates that the more unpredictable the area is, the more specific enablement is necessary. In view of the quantity of experimentation necessary, the limited working examples, the unpredictability of the art, the lack of sufficient guidance in the specification and the breadth of the claims, it would take an undue amount of experimentation for one skilled in the art to practice the claimed invention.

Applicants' arguments filed 8/21/03 have been fully considered but are not found persuasive.

Applicants' position is that (1) the term "modulating" is defined on page 4, second paragraph of the specification. (2) Claims have been amended to incorporate the limitation of wherein said fragments are selected from one or more of SEQ ID NO: 1-8. (3) As for any "autoimmune disease", the prior art studies indicate that HC gp-39 is expressed under immune conditions in which maturation occurs, indicating that potentially in all inflammatory autoimmune diseases HC gp-39 can be found (Krause et al). While HC gp-39 may not be the direct cause of the inflammation associated disease, it is the result of the localized inflammation, the induction of HC gp-39 reactive modulator cells that necessarily follows that it would be beneficial for other diseases in which the immunological activity results in expression of HC gp-39.

However, the amended claims still drawn to modulating reactivity of lymphocytes which can be stimulatory or inhibitory. The term "modulating the reactivity of lymphocytes" encompasses stimulatory and inhibitory activities, which are mutually exclusive. It is not clear if the claimed method of treating any autoimmune disease including rheumatoid arthritis is to inhibit or to stimulate the reactivity of which lymphocytes. As to items 2-3, although the specific fragments have been incorporated into the claims, the claimed method encompassed treating any autoimmune disease such as rheumatoid arthritis using any combination of HC gp-39 fragments selected from one or more SEQ ID NO: 1-8. There is no guidance and in vivo working example demonstrating that the claimed method is effective for treating any autoimmune disease by

modulating (stimulating or inhibiting) the reactivity of lymphocytes to "antigens other than HC gp-39". In fact, the specification discloses only a method of treating rheumatoid arthritis using the full-length gp-39 as tolerogen to inhibit the proliferation of T lymphocytes isolated from patient with rheumatoid arthritis. The term "modulating the reactivity of lymphocytes" is not sufficient to define the biological activity to which the HC gp-39 and fragments thereof which modulation can be inhibitory or stimulatory. Given the indefinite number of undisclosed antigen other than HC gp-39, a person of skill in the art could not predict which particular amino acid sequence of antigen other than "HC gp-39" is essential for stimulatory or which fragment is essential for inhibitory function, in turn could be used in the claimed method of treating all autoimmune disease.

7. Claims 4, 6, 10, 12 and 16 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor, at the time the application was filed, had possession of the claimed invention.

The specification does not reasonably provide a **written description** of (1) a method of treating *any* autoimmune disease such as rheumatoid arthritis by "**modulating**" the reactivity of lymphocytes associated with said disease, comprising the steps of administering a pharmaceutical composition comprising an effective amount of *any* HC gp-39 or fragments thereof wherein said fragments are selected from one or more of SEQ ID NO: 1-8 and a pharmaceutically acceptable carrier, wherein said lymphocytes are reactive to *any* "antigens other than HC gp-39" which are present in the same tissue as HC gp-39 as set forth in claim 4, (2) a method for "modulating the reactivity of lymphocytes that are reactive to *any* antigens other than HC gp-39" which are present in the same tissue as HC gp-39, comprising the steps of administering a pharmaceutical composition as set forth in claim 10, and (3) a method of treating any autoimmune disease by modulating the reactivity of lymphocytes associated with *any* undisclosed autoimmune disease comprising the steps of administering a pharmaceutical composition comprising an effective amount of HC gp-39 or fragments thereof, wherein said fragments are selected from one or more of SEQ ID NO: 1-8 and a pharmaceutically acceptable carrier, wherein said lymphocytes are reactive to *any* antigens other than HC gp-39 which are present in the same tissue as HC gp-39.

The specification discloses only a method of treating inflammatory rheumatoid arthritis by nasal induction tolerance comprising the step of administering a pharmaceutical composition



comprising an effective amount of bovine collagen type II HC gp39 (page 10) and a pharmaceutically acceptable carrier (page 14). The specification further discloses HC gp-39 induces the proliferation of PBMC in vitro obtained from some patient with rheumatoid arthritis (See page 15). The specification on page 8 discloses that administering high or low doses of the tolerogen or peptides can attain immune tolerance; the amount of tolerogen or peptide will depend on the route of administration, the time or administration, the age or the patients as well as general health conditions and diet.

With the exception of the specific full length HC gp-39 or the specific peptides that the lymphocytes reactive to in the claimed method, there is insufficient written description about the structure associated with function of (1) *any* "antigens other than HC gp-39" that the lymphocytes reactive to in the claimed method of treating (2) *any* autoimmune disease by (3) "modulating" the reactivity of any lymphocytes. Further, the term "modulating" can be inhibitory or stimulatory and these activities are mutually exclusive. The specification discloses only that the claimed method result in "down modulation of disease activity" such as inhibition of histological alterations as evidence by infiltrate score, inhibition of histological alterations as evidenced by photograph of knee joint, X-ray imaging and inhibit T cell proliferation in vitro from patient with rheumatoid arthritis, which of these activity are inhibitory. There is inadequate written description about the modulating the reactivity of lymphocytes associated with any autoimmune disease wherein the modulating is stimulatory as encompassed by the claimed method.

The term "modulating the reactivity of lymphocytes" is not sufficient to define the biological activity to which the claimed method modulates. Finally, the specification discloses only the use of HC gp-39 for treating only rheumatoid arthritis. Given the lack of a written description of *any* additional representative species of autoimmune disease using any combination of peptides in the claimed method wherein the modulating activity can be stimulatory or inhibitory, one of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species of autoimmune disease to describe the genus encompassed by the claimed method. Thus, Applicant was not in possession of the claimed genus. See *University of California v. Eli Lilly and Co.* 43 USPQ2d 1398.

Applicant is directed to the Revised Interim Guidelines for the Examination of Patent Applications Under the 35 U.S.C. 112, ¶ 1 "Written Description" Requirement, Federal Register, Vol. 66, No. 4, pages 1099-1111, Friday January 5, 2001.

Applicants' arguments filed 8/21/03 have been fully considered but are not found persuasive.

Applicants' position is that (1) the term "modulating" is defined on page 4, second paragraph of the specification. (2) Claims have been amended to incorporated the limitation of wherein said fragments are selected from one or more of SEQ ID NO: 1-8. (3) As for any "autoimmune disease", the prior art studies indicate that HC gp-39 is expressed under immune conditions in which maturation occurs, indicating that potentially in all inflammatory autoimmune diseases HC gp-39 can be found (Krause et al). While HC gp-39 may not be the direct cause of the inflammation associated disease, it is the result of the localized inflammation, the induction of HC gp-39 reactive modulator cells that is necessarily follows that it would be beneficial for other diseases in which the immunological activity results in expression of HC gp-39.

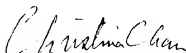
However, the amended claims still drawn to modulating reactivity of lymphocytes which can be stimulatory or inhibitory. The term "modulating the reactivity of lymphocytes" encompasses stimulatory and inhibitory activities, which are mutually exclusive. It is not clear if the claimed method of treating any autoimmune disease including rheumatoid arthritis is to inhibit or to stimulate the reactivity of which lymphocytes. The specification discloses only that the claimed method result in "down modulation of disease activity" such as inhibition of histological alterations as evidence by infiltrate score, inhibition of histological alterations as evidenced by photograph of knee joint, X-ray imaging and inhibit T cell proliferation in vitro from patient with rheumatoid arthritis, which of these activity are inhibitory. There is inadequate written description about the modulating the reactivity of lymphocytes associated with *any* autoimmune disease wherein the modulating is *stimulatory* as encompassed by the claimed method.

Although the specific fragments have been incorporated into the claims, the claimed method encompassed treating *any* autoimmune disease such as rheumatoid arthritis using any combination of HC gp-39 fragments selected from one or more SEQ ID NO: 1-8. There is inadequate written description the claimed method of treating any autoimmune disease using any combination of peptides selected from one or more of SEQ ID NO: 1-8. In fact, the specification discloses only a method of treating rheumatoid arthritis using the full-length gp-39 as tolerogen to inhibit the proliferation of T lymphocytes isolated from patient with rheumatoid arthritis. Further, the term "modulating the reactivity of lymphocytes" is not sufficient to define the

biological activity to which the HC gp-39 or fragments thereof modulate(s). With the exception of the specific full length HC gp-39 or the specific peptides that the lymphocytes reactive to in the claimed method, there is insufficient written description about the structure associated with function of *any* "antigens other than HC gp-39" that the lymphocytes reactive to in the claimed method. Given the indefinite number of "antigens other than HC gp-39", the "antigens other than HC gp-39" in the claimed method are not adequately described.

8. No claim is allowed.
9. Any inquiry concerning this communication or earlier communications from the examiner should be directed to "Neon" Phuong Huynh whose telephone number is (703) 308-4844. The examiner can normally be reached Monday through Friday from 9:00 am to 6:00 p.m. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christina Chan can be reached on (703) 308-3973. Any inquiry of a general nature or relating to the status of this application should be directed to the Technology Center 1600 receptionist (customer service) whose telephone number is (703) 872-9305.
10. Papers related to this application may be submitted to Technology Center 1600 by facsimile transmission. Papers should be faxed to Technology Center 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform to the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center telephone number is (703) 305-7401. The IFW official Fax number is (703) 872-9306. For After Final, the Fax number is (703) 872-9307.

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